

[Headache](#)

Orofacial Pain

A Guide for the Headache Physician

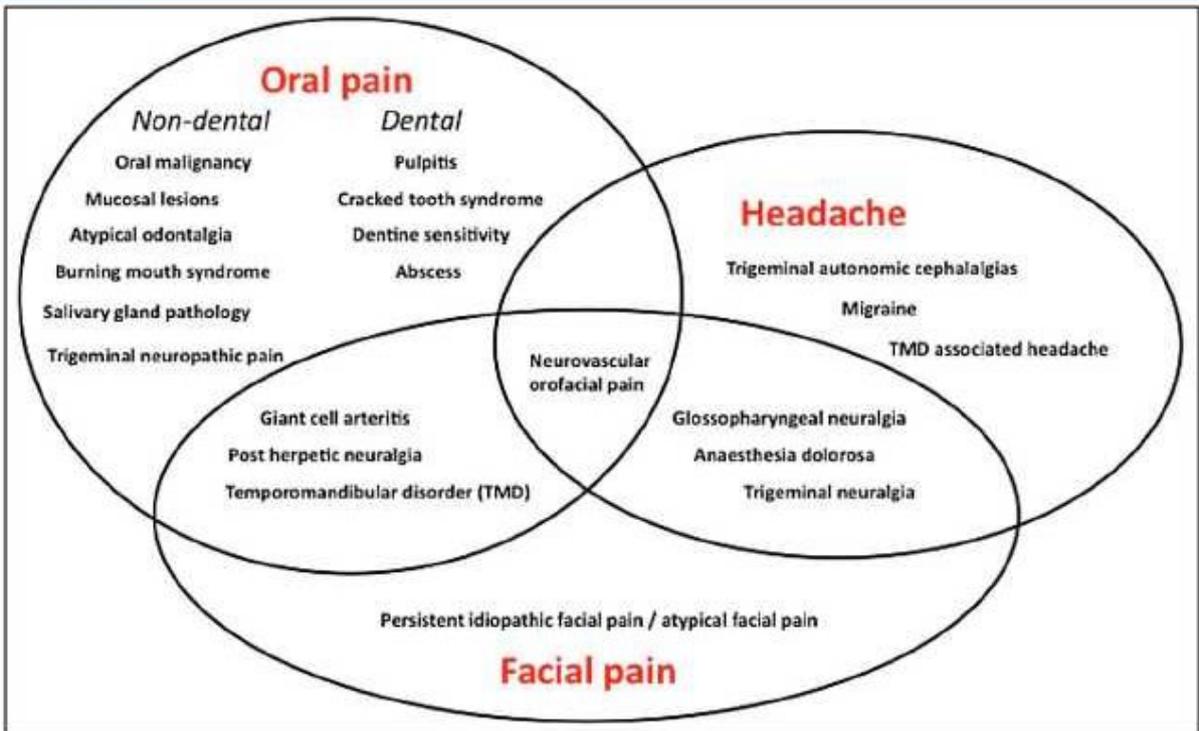
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[Disclosures](#)

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Methodology

This is a narrative, clinically orientated review of orofacial pain conditions encountered in a specialist orofacial pain clinic, with references to relevant literature. Quotations from patients are included to illustrate relevant points as these also form part of the evidence base.^[12] The types of orofacial pain have been divided into sections as shown in Figure 1. Information regarding dental causes of orofacial pain is included as this area is often unfamiliar to medical practitioners. Evidence-based management options, as far as possible, for the specific diagnoses are summarized and presented in a tabulated form ([Table](#)). In the second half of the paper, we discuss holistic management approaches to orofacial pain.



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Types of Orofacial Pain

Dental Pain

There are few causes for dental pain; however, because of significant neural convergence in the jaws and face, it may be referred, poorly localized, or misdiagnosed. The 4 major causes of dental pain are pulpitis, cracked tooth syndrome, dental abscess, and dentine sensitivity.^[13] These are often acute conditions, but because they are common, they may coexist with other chronic pains.^[14]

Both the dental pulp and periodontal ligament contain nociceptors. Nociceptive output in these areas is triggered by changes in pressure and the effect of inflammatory mediators.

Pulpitis

Pulpitis is the term used to describe pain because of inflammation of the dental pulp, and it is usually due to dental caries. Inflammation of the pulp leads to accumulation of extracellular fluid, inflammatory mediator release, and vasodilatation, which causes an elevation of pressure within the pulp chamber, which is a non-compliant space. The pressure increases further as venous stasis

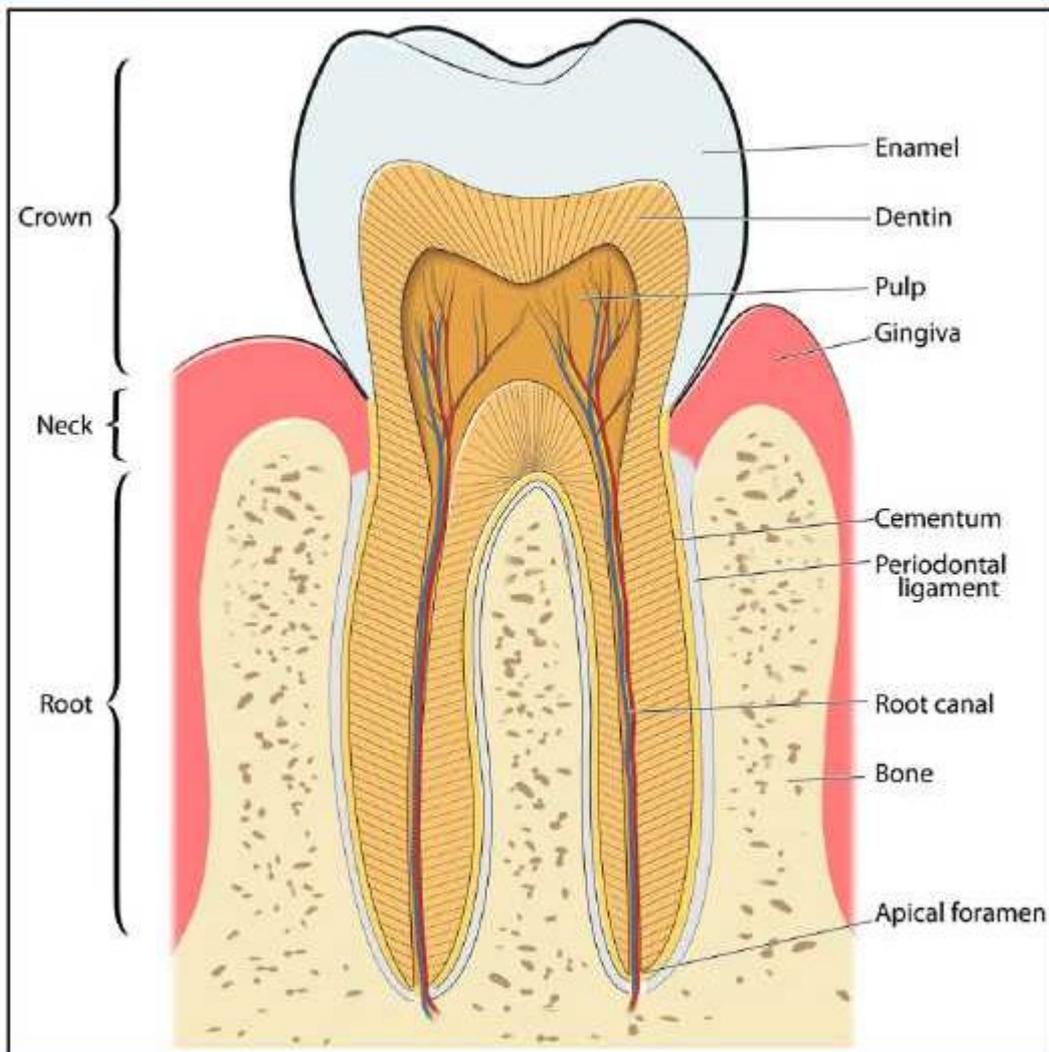
and eventually pulp necrosis occur, with release of inflammatory mediators and necrotic cell contents. Elevated pressure and inflammatory chemicals activate nociceptors in the pulp chamber causing pain.

Reversible pulpitis is defined as a transient pain in response to specific stimuli (hot, cold, sweet), which occurs when the pulp is inflamed. These symptoms resolve when the cause of the inflammation is treated. The pain of reversible pulpitis may be described as fleeting, shooting, stabbing, or sensitive.

Irreversible pulpitis is characterized by spontaneous pain, which may be worsened by or persist following the removal of a stimulus such as heat or cold. It is an indicator of incipient pulpal necrosis. The pain of irreversible pulpitis is often described as persistent, throbbing, dull, or aching. It may be worsened by physical activity and head movement.

Pulpal pain is often poorly localized as the inflammation is restricted to the pulp chamber and is thus not affecting proprioceptive nerve fibers, which are located in the periodontal ligament. It is common for patients to be unable to localize the exact source of the pain. Pulpal pain may respond to simple or opioid-based analgesics, but the pain of irreversible pulpitis will not resolve until pulpal necrosis has occurred or the pulpal tissue has been mechanically removed (by endodontic treatment).

If pulpal inflammation and infection reaches the base of the pulp chamber, an area known as the apex or root tip, it may extrude through the apical foramen into the periodontal space (Fig. 2). This will cause pain due to stimulation of nociceptors in the periodontal ligament space, and the pain will be well localized due to involvement of periodontal ligament proprioceptive fibers. Extrusion of inflammatory fluid and necrotic cell products into the periodontal space causes pain because of pressure effects, and the tooth will become exquisitely tender to touch or biting. This leads to the pain becoming very well localized, and the source of pain may be readily identified by gentle tapping on the tooth. When inflammation and infection has progressed through the apical foramen, it is described as a periapical abscess.



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Dental infection may progress into the bone, under the oral mucosa or into soft tissue spaces, and form an abscess or spreading infection, with resultant ongoing pain.

Cracked Tooth Syndrome

Cracked tooth syndrome occurs when a crack has occurred in the dental hard tissues and reaches the pulp chamber. The crack is usually not visible to the naked eye. Pain because of cracked tooth syndrome is classically intermittent, provoked on biting or releasing biting on a hard object, and is notoriously difficult to diagnose. It may be described as sharp or sensitive, and is usually related to mastication. The tooth may also become sensitive to hot and cold stimuli. It is thought that the pain is due to fluid shifts within the dentine tubules, which are generated due to pressure differences as the crack opens and closes during mastication. It can be extremely difficult to diagnose.^[15]

Dentine Sensitivity

Pain because of dentine sensitivity is classically stimulated by exposure to cold, heat, sweet foods/drinks, and mechanical trauma such as toothbrushing. The sensation is due to the movement of fluid in dentinal tubules in response to osmotic or temperature-related effects. Dentinal tubules contain the processes of cells residing in the dental pulp (odontoblasts), and fluid movement appears to trigger nociceptive output by mechanisms that are as yet unclear. Gingival recession can lead to exposure of the endings of dentine tubules, as can loss of enamel on the crown of the tooth. Dentine sensitivity is described as very rapid, fleeting, shooting pain, or sensitivity, and is always in response to an identifiable stimulus.

Non-dental Intraoral Pain

Intraoral pain may also arise from non-dental structures.^[16] Oral mucosal malignancies such as squamous cell carcinoma or salivary gland carcinoma may be painful because of ulceration or perineural invasion.

Inflammatory oral mucosal diseases such as oral lichen planus, recurrent aphthous stomatitis, vesiculobullous diseases, and oral mucosal infections such as candidiasis or herpes viruses (herpes simplex, varicella zoster) may all cause significant oral pain. Patients with hematinic deficiencies, diabetes, hematological malignancies, HIV/AIDS, and Behçet's disease may have significant oral mucosal pain and/or ulceration. Examination will usually reveal the associated oral mucosal abnormalities.^[17]

Pain may be experienced in the oral cavity, face, and neck because of salivary gland pathology. Blockage of a major salivary gland duct may be due to infection, mechanical obstruction by tumors, sialolithiasis, or ductal strictures. Obstruction of the duct will lead to pain as the gland fills with saliva, which cannot be released. Pain due to chronic ductal obstruction typically worsens preprandially or during meal times. Infection of the salivary glands will result in gland swelling, pain, and erythema/warmth of the overlying skin.

Post-Traumatic Trigeminal Neuropathic Pain/Atypical Odontalgia

This definition encompasses intraoral pain that is localized to a non-diseased dentoalveolar structure, such as a tooth or an area of alveolar ridge from which a tooth has previously been extracted.^[18] The pain is often described as "burning," "shooting," or "shock-like," and there may be significant hyperalgesia and allodynia of the affected region, often with an associated area of hypoesthesia or dysesthesia. The pain is usually

continuous, with some patients experiencing evoked severe episodes. The area is usually clearly defined with little radiation.^[19,20] Patients have described it as "nails being hit the whole time" or "kicked in the face and left bruised and burning."

Controversy remains about nomenclature and criteria for these conditions, and in this article, we differentiate them by the presence or absence of a precipitating event. It has been proposed that formal neurophysiological testing would help distinguish those with neuropathic pain compared with inflammatory causes.^[20-22] Patients with trigeminal neuropathic pain have an identifiable traumatic episode preceding the onset of the pain. The precipitating event may include physical trauma such as facial fractures, iatrogenic trauma such as restorative, endodontic, or oral surgical procedures (apicectomy, extraction, implant placement), prolonged severe infection of dentoalveolar structures, or dental procedures carried out with ineffective anesthesia.^[23] Trigeminal neuropathic pain is persistent and severe, and associated with a high level of psychological distress and a risk of further iatrogenic harm because of patients seeking ongoing dental or surgical interventions for relief of pain.

Atypical odontalgia or persistent dentoalveolar pain refers to a similar clinical presentation without a clear precipitating event.^[24,25] "Persistent dentoalveolar pain" is an ontological definition describing the symptoms and signs without attributing a causation or mechanism. Such definitions are developed using analysis of patient interviews.^[26,27] These conditions are usually managed along the same pathways as for other neuropathic pain.^[28] Until there are internationally agreed diagnostic criteria based on case-control studies and more well-conducted trials have been carried out, treatment of these conditions can vary substantially between clinicians, leaving patients confused and continually consulting in hope that a "cure" will be found.

Burning Mouth Syndrome

Burning mouth syndrome describes a collection of symptoms affecting the oral cavity, including a "burning" or painful sensation, often with an associated alteration in taste sensation and an altered perception of the quality and quantity of saliva. The symptoms are most commonly localized to the tongue.^[29,30] On clinical examination, the oral mucosa appears entirely normal. The area of abnormal sensation does not typically follow anatomic boundaries, is usually bilateral, and is continuously present. Patients may describe their symptoms as "discomfort" rather than pain. One patient described their symptoms as a "Prickly feeling like an injection wearing off," and when choosing photographic images as representative of their symptom, many choose images of fire.^[31] Other causes of oral burning sensations such as hematinic deficiencies, diabetes, other systemic diseases, and oral infections should be ruled out. The condition is most

common in perimenopausal or postmenopausal females, and is strongly associated with psychological comorbidities such as anxiety and depression.^[32] Patients often report that their symptoms are worsened during periods of psychological stress. The etiology of the condition is unclear, although recent studies have suggested the presence of a small-fiber sensory neuropathy, thus suggesting it is a form of neuropathic pain,^[33, 34] but others propose a steroid dysregulation mechanism.^[35] The condition can be difficult to manage, and a variety of RCTs have been reported, which include drug therapies and cognitive behavior therapy.^[36-38] Research on this condition is difficult to conduct in part due to its rarity and a lack of animal models; however, studies are being undertaken that indicate evidence of central changes on functional magnetic resonance imaging (MRI), thus supporting the hypothesis that there are definite neurophysiological elements to this condition, rather than it being a psychosomatic condition as has been previously suggested.

Facial Pain With/Without Intraoral Pain

TMDs

TMDs are the most common causes of orofacial pain, affecting 10–15% of the population.^[39, 40] Presenting features include pain localized to the pre- and post-auricular areas, the angle and ramus of the mandible, and the temporal region. There may be associated clicking, sticking, or locking of the temporomandibular joints. The pain may be intermittent or continuous, and is usually described as dull, aching, or throbbing, or in the words of patients: "weight on the side of the face getting heavier and heavier," "pressure feeling," "elastic band that is too tight," or "needles digging in." Some patients experience pain that is sharp or shooting in character, intermixed with dull continuous pain. The pain commonly radiates into the temporal or occipital regions into the neck and across the malar region of the face; it can be unilateral or bilateral, and of varying severity. There may be an associated bruxing or clenching habit. The pain is typically aggravated by opening the mouth wide, yawning, or chewing. There may be limitation of mouth opening.^[41]

TMD has historically been classified using the Research Diagnostic Criteria into myofascial pain, disc displacement, and other disorders,^[42, 43] as the International Classification of Headache Disorders (ICHD)-II of TMD was not useful in clinical settings.^[2] Newer classification criteria refer to myalgia, myofascial pain with referral, and myalgia with disc involvement.^[41]

A large prospective cohort study is currently underway in the USA investigating the prognostic factors related to the development of TMD.^[44-46] Participants with

and without TMD participate in a battery of psychometric, biometric, and genetic tests. Baseline data on the psychological characteristics of the TMD cases demonstrate that this population shows higher levels of distress, catastrophizing, and increased somatic awareness compared with non-TMD controls. A number of other studies have reported similar findings.^[47–49]

TMD has been linked with other psychological and chronic pain conditions, including fibromyalgia, back pain, headaches, chronic widespread pain, and hypermobility.^[50–53] Degenerative temporomandibular joint disease is rare but may occur in rheumatoid arthritis. Interest has been raised recently in the possibility of TMD-related headache, which may involve aspects of peripheral and central sensitization.^[54]

Management of TMD is primarily conservative, as in the majority of cases, the disorder is self-limiting. Careful explanations are crucial as it has been shown that patients experience a considerable amount of uncertainty both in terms of diagnosis and then management, as dentists also often find it difficult to manage.^[55–57] Approximately 10% of patients develop chronic pain, and this has been linked to fibromyalgia, depression, and chronic widespread pain.^[58] Therapies used for TMD include simple analgesia, tricyclic antidepressants, occlusal splints or bite guards, diet modifications, physiotherapy, cognitive behavioral therapy, and surgery.^[59–61] Evidence for the majority of these therapeutic options is poor, and there remains considerable confusion about the best form of management.^[7] Surgery is only indicated for TMD with significant functional limitation or in cases with associated degenerative joint disease or disc dysfunction.^[62] Education, psychological support and self-management strategies are recommended as part of a multidisciplinary approach to the management of TMD, and these should be done early to reduce costs.^[63–65] There remains considerable variation in the way TMD is diagnosed and managed partly due to conflicting evidence. It is anticipated that the large US-based Orofacial Pain: Prospective Evaluation and Risk Assessment (OPPERA) study will provide more robust evidence, as it is a prospective study that has enrolled asymptomatic participants.^[44–46]

Giant Cell Arteritis

Giant cell arteritis (GCA) is an important differential diagnosis in any patient over the age of 50 years presenting with temporal or pre-auricular pain. This condition is potentially vision-threatening and needs to be identified and treated as a matter of urgency. The pain of GCA is often described as "throbbing" and continuous, and may be associated with jaw claudication, visual symptoms, and

systemic illness, including musculoskeletal pain in the upper limbs (polymyalgia rheumatica). Clinical examination may demonstrate a reduced pulse in a tortuous temporal artery. Blood tests for erythrocyte sedimentation rate and C-reactive protein (CRP) should be performed urgently as these will assist in confirmation of the diagnosis, followed by temporal artery biopsy.^[66] If the clinical presentation is strongly suggestive of GCA, treatment with high-dose corticosteroids should be commenced prior to the receipt of test results, and urgent referral to ophthalmology should be made to avoid loss of vision.^[67]

Post-herpetic Neuralgia

Post-herpetic neuralgia is a neuropathic pain arising after reactivation of herpes virus species (most commonly varicella zoster), which may involve branches of the trigeminal nerve. The first division is most commonly affected; however, involvement of the second or third divisions may result in facial or intraoral neuropathic pain. The pain is unilateral and restricted anatomically to 1 or more branches of the trigeminal nerve, and is described as "burning" and continuous.^[18] History often reveals an episode of herpes virus reactivation including the presence of "blisters," "ulcers," or vesicles on the skin or intraorally, associated with extreme pain in the same region, which typically precedes the appearance of the vesicles. Pain may persist for up to 6 weeks following an episode of herpes virus reactivation, and allodynia is often present on examination. Ongoing pain and neurological abnormalities 3–6 months following the acute episode is classified as post-herpetic neuralgia and is common in elderly patients, resulting in considerable impact on quality of life.^[68] There is often a complaint of severe itching. Management is as for other types of neuropathic pain.^[69] However, it is important to treat the acute episode with high-dose antiviral medications and even tricyclic antidepressants in elderly patients as they are at higher risk of developing post-herpetic neuralgia. Antiviral medication should be commenced within 72 hours of the onset of rash/vesicles but may be started up to 7 days following onset, particularly in immunocompromised or older individuals.^[70]

Trigeminal Neuralgia

Trigeminal neuralgia (TN) is a condition characterized by episodic, usually unilateral, severe attacks of facial pain, which are often described as "shooting," "electric shock-like," or "stabbing." Metaphors used by patients include "plugged into the mains and switched on and off" and "rockets and explosions." The pain attacks are of very short duration (seconds) with a refractory period, and periods of complete pain remission may occur, which can last for months or even years.^[71] With time, the remission periods tend to shorten. Some patients describe a continuous dull ache or burning after an acute attack, eg, "red hot iron being pushed and turned inside the cheek," and if this sensation persists, it has variously been labeled as atypical TN, type 2 TN,^[72] or TN with concomitant pain.^[73] The pain is restricted to the anatomical boundaries of divisions of the trigeminal nerve and most commonly affects the second and third divisions. The pain is triggered by a variety of light touch stimuli, including talking, eating or tooth-brushing, face-washing, or cold winds. Patients will usually be

able to identify a discrete "trigger zone" within which sensory stimuli will produce a pain attack. It is a severe and disabling pain, and has a significant impact upon quality of life. One of our patients described her TN experience as follows: "My whole life was falling apart. My husband was losing weight, everything was falling apart in our house, my job and there was nothing I could do about the pain."

In some patients, there is an identifiable cause, such as an intracranial lesion or a vascular compression of the trigeminal nerve.^[74] TN is reported in up to 3.8% of patients with multiple sclerosis.^[75] For this reason, imaging (MRI) forms an essential part of the work-up for TN. Management will depend on whether there is an identifiable cause, but is primarily medical, and aims to achieve symptom relief. Medical management involves the use of anticonvulsants such as carbamazepine or oxcarbazepine.^[74, 76] Second line agents include lamotrigine and baclofen. Because of the increasing number of anticonvulsants now available, many patients are referred unnecessarily late for surgical interventions that can offer the best quality-of-life outcomes.^[77] Surgical procedures such as microvascular decompression may be performed if there is imaging evidence of a lesion affecting the trigeminal nerve root, and the disease is causing a significant impact on quality of life.^[78, 79] Other less invasive surgical procedures such as radiofrequency thermocoagulation, glycerol rhizotomy, balloon compression, or gamma knife surgery tend to provide shorter periods of pain relief and have a higher risk of sensory loss. They are used in patients who are medically unfit for major surgery such as microvascular decompression. It remains difficult for patients and clinicians to make decisions about treatment due to a lack of high-quality evidence. Some data suggest that many patients prefer a surgical option rather than ongoing medical management.^[80]

Glossopharyngeal Neuralgia

Glossopharyngeal neuralgia has a similar presentation to TN, although the location of the pain is different. Patients may experience paroxysmal attacks of pain felt deeply in the throat, ear, or posterior aspect of the tongue. The triggers for pain attacks include chewing, talking, drinking, and swallowing. The condition is usually managed medically with anticonvulsant drugs. Refractory cases may require surgery in the form of microvascular decompression.^[81]

Anesthesia Dolorosa

Anesthesia dolorosa is a condition arising from damage to the trigeminal nerve, usually during surgery for TN. The condition develops 3–6 months following the traumatic incident. It is characterized by "painful numbness." Patients will report continuous facial pain in an area of numbness, often described as "burning," "pressure," or "stinging." This is a typical patient description: "The right side of my face, from my chin to above my right eye, is numb and I frequently experience a 'crawling' sensation on the right side of my face and scalp. Also, my face has quite a bit of pressure and feels as though it is being pulled or tugged, as if in a visor."

The pain is persistent, severe, and associated with a high level of psychological distress and comorbidity. It is often resistant to treatment. The area involved may include all 3 divisions of the trigeminal nerve. Examination findings may include objective sensory deficits, allodynia, and hyperalgesia or hypoalgesia.^[82] Management options are as for neuropathic pain, with psychological support playing a significant role. In the new ICHD classification, this entity has been named "painful post-traumatic trigeminal neuropathy."^[18]

Persistent Idiopathic Facial Pain/Atypical Facial Pain

This term is used to describe a facial pain presentation that does not fit the clinical pattern for any other diagnosis and is relatively rare.^[18, 83] It is often continuous, "nagging" and "dull" in nature, and is not restricted by neurological anatomical boundaries.^[84, 85] An example of a patient's description of the pain is: "Concrete poured into my head and then moving around."

There is a high level of associated psychological comorbidity and a high prevalence of chronic pain elsewhere in the body.^[5, 32] It is often associated with conditions such as irritable bowel syndrome and chronic widespread pain. The etiology of the condition is unclear, although recent research has suggested the possibility of a pathophysiology similar to trigeminal neuropathic pain.^[86, 87] There is often a history of mental health problems that may predate the pain. Management is often difficult and includes medical and psychological input, using a multidisciplinary team approach.^[88-90] Because of the very broad definition that has been proposed in the new ICHD classification, this diagnosis will continue to be applied to a very heterogeneous group of patients and thus limit further research into the condition.^[18]

Headache-related Facial Pain

Migraine and Neurovascular Orofacial Pain

Migraine may manifest as facial pain either because of referral or as a phenomenon referred to as atypical or lower half migraine.^[91] Some authors have suggested the presence of a separate entity that they have named neurovascular orofacial pain (NVOP).^[92] This is a rare presentation and may mimic a number of other orofacial pain diagnoses. The pain is usually experienced in the distribution of the second or third divisions of the trigeminal nerve and is episodic. Attacks generally last for longer than 60 minutes. It is often described as "throbbing" and may have accompanying autonomic signs or systemic symptoms such as nausea. Patients may also complain of dental sensitivity, which can introduce diagnostic difficulties as patients pursue treatment for a perceived dental source of pain. NVOP has features in common with migraine as well as trigeminal autonomic cephalalgias, and it is suggested that NVOP may represent "relocated" migraine.^[93] It is important to differentiate NVOP from dental pulpal pathology, with which it is often confused due to the presence of dental sensitivity during

attacks. A case series of 7 lower facial migraines showed that all cases responded to triptans, and 3 responded to migraine prophylactic measures.^[94] Case-control studies from a range of different clinical settings are necessary in order to provide more evidence for the presence of this entity, as its management can be substantially different to other orofacial pain diagnoses.

Trigeminal Autonomic Cephalalgias

This group includes cluster headache, paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks with cranial autonomic features, and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing. These headaches are characterized by unilateral head or facial pain with cranial autonomic features that occur ipsilaterally and at the same time as the pain.^[18] Patients with these disorders may present to facial pain clinics, as the facial pain component may be more significant than the headache. Accurate history-taking is essential in formulating this diagnosis, as patients may be unaware of the autonomic symptoms unless specifically asked. Comprehensive discussion of these disorders may be found in the literature. However, more careful phenotyping and larger case series are necessary to determine which of these diagnoses are unique entities and which may represent a continuum in the natural history of these disorders.^[95,96]

TMD-Related Headache

Recent studies have described an association between TMD and headache. Many patients with TMD also report headache, and in some cases, there is a clear relationship between temporomandibular joint-related triggers and headache onset.^[97] TMD is also common among migraine and tension-type headache sufferers.^[98]

A Holistic Approach to Facial Pain

The History

Accurate and comprehensive history-taking is essential in order to gather sufficient information in order to formulate a diagnosis and treatment plan.^[99] The medical consultation has been described as "a transaction that involves translation," and further that "the physician's concern is to translate the subjective experience of illness into the recognizable discourse of medicine."^[100] It has also been suggested that we should not be "taking" a history but "receiving it."^[100] Inaccurate or inappropriate "translation" can lead to inaccuracy of diagnosis and impair the therapeutic relationship.

Our unit advocates the use of a structured or semistructured history in order to ensure consistency in history-taking and documentation, and to assist in diagnostic accuracy. An open-ended style of history-taking, rather than an interrogative approach, often yields important information and ensures that patients feel they have been listened to and their health beliefs understood.^[101]

^[102] Building a therapeutic relationship is essential in the assessment and management of chronic pain. Ensuring sufficient duration for the initial consultation, allowing the patient time to speak and express their ideas regarding the pain, and eliciting and understanding patient expectations are all essential for successful pain management.^[103] A recent study of 12 patients interviewed preconsultation and post-consultation in a pain clinic, without the knowledge of the clinicians involved, provided some of these comments:

I guess what the appointment has done is drawn a line under it and made me think, well, that's fine but nothing can be done about it so I just need to get on with things.

Even though I haven't come away with a cure, I feel in a better position to cope with my symptoms.

I felt xxx listened to me more than the other healthcare professionals I have seen and took into account the effects the pain was having on my life in general, rather than just treating me as a diagnosis.^[104]

In addition to the standard pain history, psychiatric comorbidities must be identified and addressed early in the therapeutic relationship, as they may have been present before the onset of the pain.^[15] It is also essential to elicit detailed information regarding social history, major life events, psychosocial stressors, and the impact of pain on the patient's ability to participate in the activities of daily living. Many patients with facial pain who present to secondary care or pain clinics have attended consultations with a large number of primary and secondary care providers, and may have had multiple investigations or interventions for their pain.^[7, 102, 105] This is illustrated by the following patient quotation: "A lot of people would think [that consulting a] dentist, max facs [maxillo-facial surgery], neurologist was already over the top but I wanted to be certain that I'd tried everything."

These patients often have a significant level of psychological distress, and this can impact negatively on the therapeutic relationship and management strategies. Understanding the patient's expectations and illness beliefs, and assessing negative prognostic factors such as catastrophizing or low self-efficacy

levels is essential in order to formulate an appropriate treatment plan using the biopsychosocial model that will then require a multidisciplinary team approach.^[106]

Psychological screening tools such as the National Institutes of Clinical Excellence depression screening questions, the Hospital Anxiety and Depression Scale, Patient Health Questionnaire, and the Beck Depression Inventory are useful for quantifying the degree of psychological comorbidity.^[107] The inclusion of an objective measure of pain impact on quality of life is essential in every facial pain consultation; the Graded Chronic Pain Scale, Brief Pain Inventory (including the extended version),^[108] the Pain Catastrophizing Scale, and the EuroQoL scale are useful tools. However, these measures need to be carefully interpreted in the context of the patient's comorbidities. As 1 patient commented: "And if you're very depressed and it's hard to verbalize how you feel about things, or whether you can't just mark on a scale between nought and ten what your pain is like, you know, what's your pain, is it nought or is it ten?"^[31]

Diagnostic Considerations

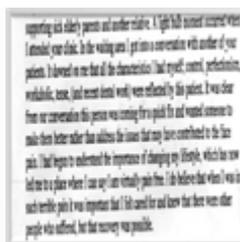
There is also the propensity for clinicians to "label" patients with a diagnosis, with the expectation that this will enable the patient to accept the condition and progress with treatment. This approach may be helpful for some patients – 1 patient stated: "I was quite relieved to have a diagnosis ... although I had hoped I would come away with a solution for a cure, I am happy now that I know the cause and that it is not serious."^[104]

However, our experience is that this is often not the case. Some facial pain presentations are diagnostically challenging, and the evolution of symptoms over time may either clarify, or rule out, the diagnosis initially given. Extant classification systems may also hinder diagnosis or result in inaccurate labeling. It has been found that the number of patients whose symptoms could not be classified as a specific diagnosis was larger in ICHD-II than in ICHD-I, with particular difficulty experienced in patients with persistent idiopathic facial pain.^[109] In a study examining the usefulness of the ICHD-II classification criteria, only 56% of patients were successfully diagnosed with orofacial pain using ICHD-II.^[2] Applying American Academy of Orofacial Pain (AAOP)/Research Diagnostic Criteria for Temporomandibular Disorders (RDCTMD) criteria, a further 37% were diagnosed with masticatory myofascial pain (MMP), and further published criteria enabled the remaining patients to be allocated to other predefined diagnoses. The authors concluded that while MMP is clearly defined by AAOP and the

RDCTMD, expansion of ICHD-II was needed so as to integrate more orofacial pain syndromes.

It may be better to give no diagnosis rather than the wrong diagnosis, as revising a diagnosis that has previously been presented to the patient as definitive can be damaging to the therapeutic relationship and the patient's confidence in the clinician. The use of a grading system such as "definite," "probable," or "possible" has been suggested for use when diagnosing neuropathic pain.^[110] This classification could be extended to other orofacial pain diagnoses as a means of managing the uncertainty in providing diagnoses for conditions that have varied clinical presentations. Ontological approaches to the diagnosis and classification of facial pain syndromes aim to reduce the problems associated with "labeling" and focus on the use of purely descriptive terms with no inferences made regarding mechanism or etiology.^[27]

"Labeling" or compartmentalizing patients into diagnostic categories also ignores the multifaceted nature of chronic pain syndromes, particularly orofacial pain. The patient is not the diagnosis – rather the pain condition has occurred in a patient who exists within a milieu of social, cultural, psychological, and cognitive influences. Patients' beliefs about their condition will also affect their disability and outcome,^[111] as the quote in Figure 3 — illustrates. Recognizing the significance of these contributory factors to the overall presentation is essential for effective therapeutic dialogue as well as good management of pain.



supporting and elderly parents and another relative. A fight had occurred several years
I attended your clinic. In the waiting area I got into a conversation with another of your
patients. I learned in one hour all the characteristics that myself, control, perfectionism,
workaholic, tense, and recent dental work were reflected by this patient. It was clear
from our conversation this person was coming for a quick fix and would not come to
make their better rather than address the issues that may have contributed to the pain
pain. That began to undermine the importance of changing my lifestyle, which has now
led me to a place where I can say I am virtually pain-free. I do believe that when I was in
such terrible pain it was important that I felt heard for and knew that there were other
people who suffered, but that recovery was possible.

[\(Enlarge Image\)](#)

Figure 3.

Extract from a patient letter about treatment of her orofacial pain.

This concept has been further explored in a recent series of qualitative studies examining patients' experience and perception of orofacial pain.^[26, 102, 105] As with any other chronic pain psychological factors will increase pain disability.^[112] Orofacial pain syndromes often co-exist with significant psychological morbidity, probably more so than other types of chronic pain,^[10, 113] and are risk factors for chronicity.^[114] Addressing these factors is essential for

appropriate management of the orofacial pain, as treatment outcome has been shown to be related to psychological comorbidity.^[49] Affective as well as interpretative and cognitive factors play an important role in the patient's perception of pain. One small qualitative study found that their patients perceived their orofacial pain to "have no limits and to repressively permeate all aspects of their existence: social, practical, and emotional."^[105] This illustrates the significant impact that orofacial pain can have on quality of life, and provides a focal point for assessment of pain management outcomes. Patients need to know that although the sensation of pain may not be completely alleviated by treatment, the impact of pain upon their daily life can certainly be modulated.

Management

Chronic pain management should be holistic in nature and approach, and involves addressing all the factors that modulate the pain experience.^[7] Addressing unrealistic patient expectations is important for setting achievable treatment goals. There remains a common perception that pain should always be curable, as demonstrated in this quote from a patient: "Many don't understand the pain I feel. They think I should be over this pain by now. Others feel I should seek other doctors. They feel there should be something to relieve this terrible pain and ask me why I'm not trying to find it, if it is so bad."

Pain as defined by International Association for the Study of Pain is both a "sensory and emotional experience," and it should be managed as such. A recent study has shown that chronic musculoskeletal pain can be experienced as a "constant adversarial struggle," and the researchers suggest that patient and clinician expectations of a diagnosis and cure need to be challenged.^[115] Beliefs, coping strategies, and catastrophizing predict functioning in patients with chronic pain, and this should be considered when individualizing pain management programs.^[116] This extends to patients' beliefs about medication as these will influence adherence.^[117]

Successful pain management is also related to the patient's self-efficacy beliefs and ability to learn and use positive coping strategies.^[118] Recognition of the contribution of social, psychological, and lifestyle factors to the pain experience, as expressed in the patient quote earlier, is essential for taking the next steps in chronic pain management and achieving a reduction in the impact of pain on quality of life. The provision of support for these next steps is a fundamental part of multidisciplinary pain management. Pain management programs delivered in group settings normalizes the pain experience, and the concept of an improved

pain experience because of observation of others with a similar complaint is also expressed by the patient quoted earlier. Newer techniques such as an e-learning cognitive behavioral therapy model can be helpful.^[119] Supported but self-directed pain management focused around lifestyle change and reducing the impact of pain on quality of life is a more effective management approach in chronic pain than the traditional, didactic biomedical model, but these strategies need to be tailored appropriately.^[120]

Conclusion

Orofacial pain in its fullest definition affects up to a quarter of the population, and the associated morbidity, social impact, and health costs can be high if these conditions are not accurately diagnosed and managed in a timely fashion. Recognition of the significant contribution and high prevalence of psychological distress and comorbidity is essential for successful management. Multidisciplinary approaches and a biopsychosocial model of pain management are an essential adjunct to established evidence-based medical and surgical management of these conditions.